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 NEWS 24  AUG 15  CAplus currency for Korean patents enhanced
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                  enhanced for more flexible patent number searching
 NEWS 26  AUG 27  CAS definition of basic patents expanded to ensure
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FILE 'BIOSIS' ENTERED AT 15:27:53 ON 16 SEP 2008
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=> Ox40
L1        1031 OX40

=> DNA vaccine
L2       10025 DNA VACCINE

=> L1 and l2
L3           4 L1 AND L2

=> immunogenic (L) polypeptide
L4        1748 IMMUNOGENIC (L) POLYPEPTIDE

=> L1 and L4
L5           0 L1 AND L4

=> fusion (s) protein
L6      120827 FUSION (S) PROTEIN

=> L6 and L1
L7          94 L6 AND L1
```

=> HSV
L8 30053 HSV

=> L8 and L7
L9 6 L8 AND L7

=> D L9 IBIB ABS 1-6

L9 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2007:1091580 CAPLUS
DOCUMENT NUMBER: 148:353490
TITLE: Inhibition of OX40-Ig on herpetic stromal keratitis in murine model
AUTHOR(S): Xia, Likun; Chen, Xiaolong; Zhu, Yingming; Zhou, Jing
CORPORATE SOURCE: Department of Ophthalmology, Affiliated Second Hospital, China Medical University, Shenyang, 110004, Peop. Rep. China
SOURCE: Yanke Yanjiu (2006), 24(5), 479-483
CODEN: YAYAFH; ISSN: 1003-0808
PUBLISHER: Henan Institute of Ophthalmology
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB Herpetic stromal keratitis (HSK) is an immunoinflammatory lesion in the cornea of the eye set off by the infection with HSV-1. The disease appears to be orchestrated by CD4+ T cells. In current study, it was investigated that the inhibition of OX40-Ig on the inhibition of HSK. Corneas of right eyes from 90 BALB/c mice were infected with 106 PFU of HSV-1 McKrae strain. Mice were injected i.p. with OX40-Ig or IgG Fc or PBS given on day 0, 2, 4 after the infection. CD4+ T cells from peripheral blood of mice were analyzed on FACS 440 analyzer. The clin. evaluations of infected eyes were taken under the slit-lamp microscope, and the histol. changes of corneas were observed under the optical microscope. Virus titers in corneas after HSV-1 infection were tested with VERO cells, and delayed type hypersensitivity was observed The effects of OX40-Ig on HSK were evaluated. As measured by flow cytometry, in the mice treated with OX40-Ig, 78.2% of CD4+ T cells were reduced. 83.3% Of the HSV-1-infected control mice developed severe stromal keratitis, but only 20.0% of mice treated by OX40-Ig developed HSK. Lesions in OX40-Ig treated mice showed markedly reduced severity by slit-lamp microscope, and histol. the corneal stroma had a decrease in inflammatory cell infiltration compared to the control group, and the delayed type hypersensitivity was reduced. The results provide an evidence that blockade of OX-40/OX-40L co-stimulation by OX40-Ig can inhibit the proliferation of CD4+ T cells and impair onset and severity of HSK.

L9 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2007:254551 CAPLUS
DOCUMENT NUMBER: 146:294007
TITLE: Expression and function of the OX40/OX40L costimulatory pair during herpes stromal keratitis
AUTHOR(S): Lepisto, Andrew J.; Xu, Min; Yagita, Hideo; Weinberg, Andrew D.; Hendricks, Robert L.
CORPORATE SOURCE: Department of Ophthalmology, School of Medicine, University of Pittsburgh, Pittsburgh, PA, USA
SOURCE: Journal of Leukocyte Biology (2006), Volume Date 2007, 81(3), 766-774
CODEN: JLBIE7; ISSN: 0741-5400
PUBLISHER: Federation of American Societies for Experimental

Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Herpes stromal keratitis (HSK) is an immunopathol. disease regulated by Th1 CD4 T cells, which require APC and costimulation within the infected cornea to mediate disease. Recent studies suggest the OX40: OX40 ligand (OX40L) interaction enhances effector cell cytokine secretion at inflammatory sites. OX40+ cells were detected in HSV-1-infected mouse corneas as early as 3 days postinfection (dpi), prior to the onset of HSK, and their frequency increased through 15 dpi, when all mice exhibited severe HSK. OX40L+ cells were first detected at 7 dpi, coincident with the initiation of HSK. It is interesting that the OX40L+ cells did not coexpress MHC class II or the dendritic cell (DC) marker CD11c. The authors' findings demonstrate rapid infiltration of activated (OX40+) CD4+ T cells into HSV-1-infected corneas and expression of OX40L on MHC class II-neg. cells but surprisingly, not on MHC class II+ CD11c+ DC, which are present in the infected corneas and required for HSK. Moreover, neither local nor systemic treatment of mice with a blocking antibody to OX40L or with a blocking fusion protein altered the course of HSK, possibly as a result of a lack of OX40L expression on functional APC.
REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:679028 CAPLUS
DOCUMENT NUMBER: 141:409506
TITLE: Anti-tumor therapeutic efficacy of OX40L in murine tumor model
AUTHOR(S): Ali, Selman A.; Ahmad, Murrium; Lynam, June; McLean, Cornelia S.; Entwisle, Claire; Loudon, Peter; Choolun, Esther; McArdle, Stephanie E. B.; Li, Geng; Mian, Shahid; Rees, Robert C.
CORPORATE SOURCE: School of Science, Nottingham Trent University, Nottingham, NG11 8NS, UK
SOURCE: Vaccine (2004), 22(27-28), 3585-3594
CODEN: VACCDE; ISSN: 0264-410X
PUBLISHER: Elsevier Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB OX40 ligand (OX40L), a member of TNF superfamily, is a co-stimulatory mol. involved in T cell activation. Systemic administration of mOX40L fusion protein significantly inhibited the growth of exptl. lung metastasis and s.c. established colon (CT26) and breast (4T1) carcinomas. Vaccination with OX40L was significantly enhanced by combination treatment with intra-tumor injection of a disabled infectious single cycle-herpes simplex virus (DISC-HSV) vector encoding murine granulocyte macrophage-colony stimulating factor (mGM-CSF). Tumor rejection in response to OX40L therapy required functional CD4+ and CD8+ T cells and correlated with splenocyte cytotoxic T lymphocytes (CTLs) activity against the AH-1 gp70 peptide of the tumor associated antigen expressed by CT26 cells. These results demonstrate the potential role of the OX40L in cancer immunotherapy.
REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 4 OF 6 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
ACCESSION NUMBER: 2007:226155 BIOSIS
DOCUMENT NUMBER: PREV200700227511
TITLE: Expression and function of the OX40/OX40L

costimulatory pair during herpes stromal keratitis.
AUTHOR(S): Lepisto, Andrew J.; Xu, Min; Yagita, Hideo; Weinberg, Andrew D.; Hendricks, Robert L. [Reprint Author]
CORPORATE SOURCE: Eye and Ear Inst Pittsburgh, 203 Lothrop St,Room 922, Pittsburgh, PA 15213 USA
hendricksrr@upmc.edu
SOURCE: Journal of Leukocyte Biology, (MAR 2007) Vol. 81, No. 3, pp. 766-774.
CODEN: JLBIE7. ISSN: 0741-5400.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Apr 2007
Last Updated on STN: 4 Apr 2007
AB Herpes stromal keratitis (HSK) is an immunopathological disease regulated by Th1 CD4 T cells, which require APC and costimulation within the infected cornea to mediate disease. Recent studies suggest the OX40:OX40 ligand (OX40L) interaction enhances effector cell cytokine secretion at inflammatory sites. OX40(+) cells were detected in HSV-1-infected mouse corneas as early as 3 days postinfection (dpi), prior to the onset of HSK, and their frequency increased through 15 dpi, when all mice exhibited severe HSK. OX40L(+) cells were first detected at 7 dpi, coincident with the initiation of HSK. It is interesting that the OX40L(+) cells did not coexpress MHC Class II or the dendritic cell (DC) marker CD11c. Our findings demonstrate rapid infiltration of activated (OX40(+)) CD4(+) T cells into HSV-1-infected corneas and expression of OX40L on MHC Class II-negative cells but surprisingly, not on MHC Class II+ CD11c(+) DC, which are present in the infected corneas and required for HSK. Moreover, neither local nor systemic treatment of mice with a blocking antibody to OX40L or with a blocking fusion protein altered the course of HSK significantly, possibly as a result of a lack of OX40L expression on functional APC.

L9 ANSWER 5 OF 6 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:452715 BIOSIS
DOCUMENT NUMBER: PREV200400449410
TITLE: Anti-tumour therapeutic efficacy of OX40L in murine tumour model.
AUTHOR(S): Ali, Selman A.; Ahmad, Murrium; Lynam, June; McLean, Cornelia S.; Entwisle, Claire; Loudon, Peter; Choolun, Esther; McArdle, Stephanie E. B.; Li, Geng; Mian, Shahid; Rees, Robert C. [Reprint Author]
CORPORATE SOURCE: Sch Sci, Nottingham Trent Univ, Clifton Lane, Nottingham, NG11 8NS, UK
robert.rees@ntu.ac.uk
SOURCE: Vaccine, (September 9 2004) Vol. 22, No. 27-28, pp. 3585-3594. print.
ISSN: 0264-410X (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Nov 2004
Last Updated on STN: 24 Nov 2004
AB OX40 ligand (OX40L), a member of TNF superfamily, is a co-stimulatory molecule involved in T cell activation. Systemic administration of mOX40L fusion protein significantly inhibited the growth of experimental lung metastasis and subcutaneous (s.c.) established colon (CT26) and breast (4T1) carcinomas. Vaccination with OX40L was significantly enhanced by combination treatment with intra-tumour injection of a disabled infectious single cycle-herpes simplex virus (DISC-HSV) vector encoding murine granulocyte macrophage-colony stimulating factor (mGM-CSF). Tumour rejection in

response to OX40L therapy required functional CD4+ and CD8+ T cells and correlated with splenocyte cytotoxic T lymphocytes (CTLs) activity against the AH-1 gp70 peptide of the tumour associated antigen expressed by CT26 cells. These results demonstrate the potential role of the OX40L in cancer immunotherapy.

L9 ANSWER 6 OF 6 BIOSIS
ACCESSION NUMBER: 2002:38303 BIOSIS
DOCUMENT NUMBER: PREV200200038303
TITLE: Combined experimental anti-tumour therapy using a DISC-HSV delivery system for mGM-CSF and OX40 ligand.
AUTHOR(S): Rees, Robert C. [Reprint author]; Ali, S. A.; Lynam, J.; McLean, C. S.; Choolun, E.; Entwisle, C.
CORPORATE SOURCE: Cantab Pharmaceuticals Research Ltd, Cambridge, UK
SOURCE: Proceedings of the American Association for Cancer Research Annual Meeting, (March, 2001) Vol. 42, pp. 818-819. print.
Meeting Info.: 92nd Annual Meeting of the American Association for Cancer Research. New Orleans, LA, USA. March 24-28, 2001.
ISSN: 0197-016X.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 2 Jan 2002
Last Updated on STN: 25 Feb 2002

=> D L3 IBIB ABS 1-4

L3 ANSWER 1 OF 4 CAPLUS
ACCESSION NUMBER: 2007:859817 CAPLUS
DOCUMENT NUMBER: 147:298670
TITLE: Enhanced protective efficacy and reduced viral load of foot-and-mouth disease DNA vaccine with co-stimulatory molecules as the molecular adjuvants
AUTHOR(S): Xiao, Chong; Jin, Huali; Hu, Yanxin; Kang, Youmin; Wang, Junpeng; Du, Xiaogang; Yang, Yu; She, Ruiping; Wang, Bin
CORPORATE SOURCE: State Key Laboratory for Agro-Biotechnology, Key Laboratory of Agro-Microbial Resources and Applications of MOA, China Agricultural University, Beijing, 100094, Peop. Rep. China
SOURCE: Antiviral Research (2007), 76(1), 11-20
CODEN: ARSRDR; ISSN: 0166-3542
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB To improve efficacy of DNA vaccination, various approaches have been developed, including the use of plasmid expressing co-stimulatory mols. as mol. adjuvants. Here, the authors investigated whether co-inoculation of a construct expressing either 4-1BBL or OX40L as the mol. adjuvant with FMDV DNA vaccine, pcD-VP1, can increase immune responses and protective efficacies. Compared to the group immunized with pcD-VP1 alone, the co-inoculation of either mol. adjuvant induced a higher ratio of IgG2a/IgG1, higher levels of expression of IFN-γ in CD4+ and CD8+ T cells and antigen-specific CTL responses, and more importantly provided an enhanced protection against the live FMDV challenge in animals. Concurrently, 4-1BBL as the mol. adjuvant dramatically reduced the viral loads of FMDV in vivo after the challenge. Thus, co-stimulatory

mols. 4-1BBL and OX40L can enhance the antigen-specific cell-mediated responses elicited by VP1 DNA vaccine and provide an enhanced protective efficacy with the reduced viral loads.

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2004:1156439 CAPLUS
DOCUMENT NUMBER: 142:73408
TITLE: DNA vaccines comprising immunomodulatory proteins and antigen from pathogens
INVENTOR(S): Weiner, David B.; Muthumani, Karuppiah; Kutzler, Michele; Choo, Andrew K.; Chattergoon, Michael A.
PATENT ASSIGNEE(S): The Trustees of the University of Pennsylvania, USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2004112706	A2	20041229	WO 2004-US19028		20040614
WO 2004112706	A3	20050414			
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW					
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
AU 2004249191	A1	20041229	AU 2004-249191		20040614
CA 2529051	A1	20041229	CA 2004-2529051		20040614
EP 1633372	A2	20060315	EP 2004-755303		20040614
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK					
JP 2007502868	T	20070215	JP 2006-533794		20040614
US 20070104686	A1	20070510	US 2004-560653		20040614
PRIORITY APPLN. INFO.:			US 2003-478187P	P	20030613
			US 2003-478230P	P	20030613
			US 2003-478250P	P	20030613
			WO 2004-US19028	W	20040614

AB The authors disclose the use of recombinant vaccines and live attenuated pathogens comprising one or more isolated nucleic acid mols. that encode an immunogen in combination with an isolated nucleic acid mol. that encodes an immunomodulator protein selected from the group consisting of: Fos, c-jun, Sp-1, AP-1, AP-2, p38, p65Rel, MyD88, IRAK, TRAF6, IκB, inactive NIK, SAP kinase, SAP-1, JNK, interferon response genes, NF-κB, Bax, TRAIL, TRAIL receptors, DcR5, TRAIL-R3, TRAIL-R4, RANK, RANK ligand, Ox40, Ox40 ligand, NKG2D, MICA, MICB, NKG2A, NKG2B, NKG2C, NKG2E, NKG2F, TAP1, TAP2 and functional fragments thereof.

L3 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2001:313168 CAPLUS
TITLE: Papers to Appear in Forthcoming Issues

AUTHOR(S): Anon
SOURCE: Cellular Immunology (2001), 208(2), 148
CODEN: CLIMB8; ISSN: 0008-8749
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB (Received and Accepted Dates Follow Title)Mice Disrupted for the KvLQT1 Potassium Channel Regulator IsK Gene Accumulate Mature T Cells. Dominique Chabannes, Jacques Barhanin, and Denis Escande. (Received 9/27/00; accepted 3/7/01.)Pos. and Neg. Consequences of Soluble Fas Ligand Produced by an Antigen-Specific CD4+ T Cell Response in Human Carcinoma Immune Interactions. Elke S. Bergmann-Leitner and Scott I. Abrams. (Received 12/18/00; accepted 3/7/01.)Mol. Cloning and Expression Pattern of Porcine Myeloid DAP12-Associating Lectin-1. Daesong Yim, Hyun-Bae Jie, John Sotiriadis, Yoon-Sang Kim, and Yoon B. Kim. (Received 12/13/00; accepted 3/4/01.)OX40 Ligation Enhances Cell Cycle Turnover of Ag-Activated CD4 T Cells in Vivo. Amy R. Weatherill, Joseph R. Maxwell, Chikara Takahashi, Andrew D. Weinberg, and Anthony T. Vella. (Received 1/23/01; accepted 3/10/01.)Codelivery of DNA Coding for the Soluble Form of CD86 Results in the Down-Regulation of the Immune Response to DNA Vaccines. Juan Flo, Sergio Tisminetzky, and Francisco Baralle. (Received 10/23/00; accepted 3/18/01.)Dendritic Cells Issued in Vitro from Bone Marrow Produce PGE2 That Contributes to the Immunomodulation Induced by Antigen-Presenting Cells. H. Harizi, M. Juzan, C. Grosset, M. Rashedi, and N. Gualde. (Received 11/24/00; accepted 3/15/01.)A "Chimeric" C57L-Derived Ly49 Inhibitory Receptor Resembling the Ly49D Activation Receptor. Indira K. Mehta, Hamish R. C. Smith, Jian Wang, David H. Margulies, and Wayne M. Yokoyama. (Received 1/17/01; accepted 3/14/01.)Idiotypic-Anti-idiotypic B Cell Interactions Generated against a Protective Antigen of a Morbillivirus in Mice. Shibani Mitra-Kaushik, M. S. Shaila, Anjali Karanade, and Rabindranath Nayak. (Received 10/16/00; accepted 3/22/01.). (c) 2001 Academic Press.

L3 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1998:684978 CAPLUS
DOCUMENT NUMBER: 129:274700
ORIGINAL REFERENCE NO.: 129:56017a,56020a
TITLE: DNA encoding targeting protein fused to antigen or epitope in enhancement of immune response to DNA vaccines
INVENTOR(S): Boyle, Jefferey Stephen; Brady, Jamie Louise; Lew, Andrew Mark
PATENT ASSIGNEE(S): The Council of the Queensland Institute of Medical Research, Australia; Commonwealth Scientific and Industrial Research Organisation; The University of Melbourne; The Walter and Eliza Hall Institute of Medical Research; CSL Ltd.
SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9844129	A1	19981008	WO 1998-AU208	19980326

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT,

UA, UG, US, UZ, VN, YU, ZW
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

CA 2285692	A1	19981008	CA 1998-2285692		19980326
AU 9864902	A	19981022	AU 1998-64902		19980326
AU 728962	B2	20010125			
EP 972054	A1	20000119	EP 1998-910530		19980326
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI					
NZ 500151	A	20010126	NZ 1998-500151		19980326
JP 2001522235	T	20011113	JP 1998-540989		19980326
ZA 9802608	A	19981008	ZA 1998-2608		19980327
US 20030035793	A1	20030220	US 2002-185318		20020628
US 7423016	B2	20080909			
US 20030072742	A1	20030417	US 2002-185799		20020628
US 7423023	B2	20080909			
CA 2489940	A1	20060608	CA 2004-2489940		20041208
PRIORITY APPLN. INFO.:			AU 1997-5891	A	19970327
			AU 1998-1830	A	19980213
			WO 1998-AU208	W	19980326
			US 2000-402020	A1	20000328

AB The present invention provides methods of enhancing the immune response to an immunogen and to compns. for use in these methods. In particular the present invention provides a DNA mol. for use in raising an immune response to an antigen. The DNA mol. includes a first sequence encoding a targeting mol., a second sequence encoding the antigen or an epitope thereof, and optionally a third sequence encoding a polypeptide which promotes dimerization or multimerization of the product encoded by the DNA mol. Immunization of mice with a number of DNA sequences encoding CTLA4-antigen fusions enhanced the immune response to the antigen.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT